actly corresponded with this case. The involvement of the lower legs is not so unusual, but in this case simply the muscles below the thigh were involved. Atrophy of the muscles of the thigh is common in progressive muscular dystrophy. He thought we should distinguish between the Charcot-Marie-Tooth and the Landouzy-Dejerine type on account of the difference in pathology; one is a disease of the nerve, while the other is pure myotrophia. On that account we should not try to connect the two syndromes. Unless the family history be carefully investigated, the disease might be hereditary without being known to be so from the statements of the patient.

HEREDITARY CEREBELLAR ATAXIA.

Dr. M. Mailhouse presented a patient, F. F., who was admitted to the New Haven Hospital, January 13, 1900, with the following history: Age, 32; single; birthplace, United States; occupation, postal clerk; father and mother living and well; has five sisters living, and all in good health. One sister died of pulmonary tuberculosis, and another died in infancy; mother's mother also died of pulmonary tuberculosis. A maternal aunt is at present an inmate of the Connecticut Asylum for the Insane. Mother and father have had muscular rheumatism. The patient had scarlet fever and diphtheria at fifteen years of age. When a boy he had rheumatism of mild degree. In 1887 had sunstroke. No syphilis, alcoholism or tuberculosis. The history taken on admission to the hospital states that the patient first noticed in 1887 a dragging of the toes on walking, and that the shoes curled up and were worn at the toes. He did not walk with a stamping gait, and could direct the feet properly. The legs did not cross in walking. On closing the eyes while standing he was unable to keep his balance. Had no pain in the legs but considerable backache. Then the hands began to tremble (1888) and the legs shook on walking. These symptoms increased so that he had to use two canes in walking in 1888; in 1889 he used a crutch and one cane, and in 1890, two crutches. Had lost 57 pounds in weight within the past ten years. Has headaches off and on, but they are not severe. He states that when twenty-one years of age, he noticed an inability to run; he could walk, but when he ran "his legs got mixed up as though they were playing cross-tag with one another." For the past four years erections have been incomplete. At one time he had frequent urination.

He is a man above medium height, of good frame, but somewhat emaciated. As he stands supported by two crutches, the nodding tremor of the head, which is aggravated by walking, is very noticeable.

There is marked asymmetry of the head, a flattening of the occiput, and pronounced sloping of the forehead, with considerable flattening over left parietal region. There is some facial asymmetry also, as is evidenced by a greater prominence of the right supraorbital arch than of the left. The nose is directed toward the right, and the left palpebral fissure is smaller than the right. The gait is markedly ataxic, but he is able to get about fairly well on a level with the aid of crutches, but loses his balance readily on attempting to ascend a step. There is absolutely no muscular weakness in his legs. The knee-jerks are present, but not exaggerated. There is no ankle-clonus. The Romberg symptom is present, and he is supported with difficulty when he stands with closed eyes. There is no impairment of any form of sensation. The movements are performed quickly, and are jerky. The ataxia of the upper extremities is much less evident than in the lower. The speech is noticeably affected, but changes, being at times scanning or staccato, and at other times characterized by indecision and unsteadiness in the muscles of vocal expression; it is decidedly an ataxic speech.

The pupils react neither to light nor during accommodation. There is marked lateral nystagmus, and on looking upward a rotatory nystagmus; when the eyes are at rest there is no oscillation. There are no contractures and no scoliosis. The grasp of the hands is strong and the muscles of the arms show no loss of power. The pectoral muscles are wasted, otherwise no atrophies are present. There is no optic atrophy and no limitation of the visual fields, but he is myopic, requiring a lens of seven diopters.

The superficial reflexes are present, the plantar being very active. Urine presents no abnormality. The handwriting is like that of a tabetic.

A review of the symptoms reveals the marked ataxia of the whole muscular system without any paralytic phenomena. The case resembles Friedreich's ataxia in its mode of development and general appearance, but differs from the typical disease in its later onset, in retaining the knee-jerks, in the late and mild affection of the arms, in the loss of the ocular reflexes, and in the absence of contractures or paralysis. The man has been afflicted twelve years, and there is no paralysis or contracture present. The question might arise as to the case being a multiple sclerosis, but this affection could be ruled out by the mode of onset, by the normal reflexes and sensory phenomena, and the absence of intention tremor. Furthermore the long duration of the illness without any paralyses, ocular or corporal, militated against such a diagnosis.

Dr. Caswell said that in the form of ataxia described, the fundus oculi is generally affected and the knee-jerks are increased; in this way discrimination is made between cerebellar and ordinary ataxia.

Dr. Fisher thought that the sunstroke may have had something to do with the symptoms. He remembered a case of sunstroke followed by symptoms of this type, resembling multiple sclerosis, not conforming strictly to it. Taking that as an etiological factor, we have ordinarily some meningitis, some change affecting the cortex of the brain and producing ataxic symptoms; we see ataxia, inco-ordination of the muscles, spastic condition of the patient,—a degenerative condition secondary to some such lesion as might be caused by the effects of the sun.

Dr. Fraenkel thought that there was a great difference between cerebellar ataxia and ataxia produced by disease of any other parts of the co-ordinatory system.

Dr. Peterson said he had examined the man before the meeting and thought the diagnosis lay between multiple sclerosis and cerebellar hereditary ataxia. He was inclined to think Dr. Mailhouse was right in his diagnosis of cerebellar hereditary ataxia on account of the pupillary condition; no reaction of light,—and because he had more of an ataxia than tremor of the ordinary multiple sclerosis type.

Dr. Wm. B. Pritchard asked whether there were anything of note in the subjective history of this case as regards vision, hearing, or persistence of vertigo or headaches that added to the data.

Dr. Mailhouse replied that there were no subjective symptoms at all, except backache and difficulty of manipulating the legs. He was strongly myopic without optical strain.

RAYNAUD'S DISEASE OF A GANGRENOUS TYPE.

Dr. Wm. H. Porter presented this case: The man came to this country some years ago; for a year or so was a hod carrier, and then went into an iron foundry where he has worked since. Has a very pronounced alcoholic habit, and uses tobacco to a considerable extent. In the middle of April he noticed that the fingers of both hands were very white and icy cold. This lasted for two weeks and was followed by superficial gangrene, discoloration and blackness of the four fingers of both hands, a little discoloration of the left ear, and a spot on the opposite ear. This condition lasted for ten days before he came to the hospital. He suffered intolerable pain and got very little sleep. The urine showed slight traces of albumin, a decrease in urea and pronounced amount of indican.

The patient had a very high pulse and he was given doses of nitroglycerine with strychnine and caffein to improve the circulation. Lead and opium dressing was applied and gave relief.